"The cases are, after all, examples of lymphosarcoma, excepting that the type of the new tissue differs much from lymphoid structures and agrees better with what is usually denominated mixed-cell sarcoma. In the review of the literature which I made four or five years ago I do not remember to have found similar cases described. I do not, however, see that any other designation than Hodgkin's disease, or one of its synonyms, is applicable to the symptom-complex of this disease, and, therefore, this name, or lymphosarcoma, must be applied to the pathological process."

A CASE OF ACUTE GANGRENOUS PANCREATITIS, WITH FAT NECROSIS.[1]

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Since the monograph of Fitz[1] on "Acute Pancreatitis" was published in 1889 considerable careful clinical observation has been made upon pancreatic diseases. During the last twelve months three important contributions have been made to the subject, and the list of reported cases considerably increased. Korte,[2] in the *Deutsche Chirurgie*, Lieferung, 45 D., has published a monograph, with special reference to the surgical aspect of pancreatic diseases. In Nothnagel's *Special Pathologie und Therapie*, Bd. xviii., Oser[3] has written a comprehensive article, with full bibliography. In January of this year Lancereaux,[4] in his treatise upon diseases of the liver and pancreas, gives a complete account of all pancreatic diseases.

I desire to report the following case of pancreatic disease because of the comparative rarity of the condition, and the presence of some rather unusual symptoms.

Mrs. A. B. G., Hospital No. 1097, aged sixty-four years, white, married, was admitted into the private ward of the Pennsylvania Hospital August 1, 1898. Her father and a sister had died with chronic renal disease. Her mother had always been healthy, and died of senility. The patient's personal history was as follows: She was an active worker in charitable and philanthropic societies; her general health had been good, but during the last ten years of her life she had been rather a frequent sufferer with gastro-intestinal attacks, which were characterized by pain and distention of the stomach. Her physician has told me that she did not seem able to digest starchy foods. These attacks would, however, pass off in a few days' time, when she would be as well as usual. In 1895-96 she had an illness, during a portion of which she was a patient at the German Hospital. This sickness was characterized by constant pricking sensations over the entire body, but especially marked about the neck, shoulder, and trunk She became much emaciated, and did not recover until she tried a change of climate, going to Glenwood Springs, Colorado, and taking medicinal baths at that place. It seems to me that this must have been a neurosis, and had no connection with her subsequent illness. Prior to the present sickness she had been working extremely hard, her digestion had not been good, and she seemed rather mentally depressed. For a week or ten days before her sickness she was obstinately constipated, with swelling of the abdomen and some flatulence. The present attack began suddenly, about half-past eleven at night, between four and five hours after the ingestion of food. Following her supper, which she had about six o'clock, she spent the evening with some friends; shortly after going to bed she was seized with a violent pain in the pit of the stomach; the abdomen became tender and swollen; she belched large quantities of gas and vomited copiously. Her attending physician administered morphine that night, and again on the following day, and the pain was considerably allayed. The bowels, which had not been moved for six or seven days, now became loose, and were moved almost constantly, the stools being dark and rather liquid. Twenty-four hours after her illness began she became delirious, or, as her physician said, went into coma. On the fifth day of her illness she was sent to the hospital. Upon examination her condition was as follows:

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[1] Read before Medical Section, College of Physicians, May 8, 1899.

She was a well-nourished woman, weighing probably 160 pounds. She was very restless, constantly shifting her position. Her temperature was 102.2°; pulse 88, soft, rather small, irregular, and intermittent; respiration 24. Her mental condition was interesting; she seemed to understand questions asked, and answered them in a coherent manner, but would then begin to talk or sing to herself. It was, therefore, not possible to assure one's self that her replies were accurate. Her condition resembled a mild mania. Her tongue was moist, with a brownish coat. There was no jaundice present; the pupils were contracted to almost pin-point size, and so continued. The heart's action was irregular and intermittent; abortive systoles were frequent. At the apex the first sound was impure; no murmur was heard. The belly was tightly distended with gas. The skin was red, evidently from counter irritation. On palpation at this time no tenderness was elicited in the epigastric region; no tumor or mass was felt. The liver and spleen were normal in percussion area. The urine, which always had to be withdrawn by catheter, was diminished in amount and continued to be so—twenty-six ounces in the first twenty-four hours; it was light-yellow, 1012, containing neither albumin nor sugar, but microscopic examination showed many hyalo-granular, dark granular, and fat casts, and a few leucocytes. At one subsequent examination there was a suspicion that sugar was present. Subsequently a trace of albumin was found, with a large number of dark granular casts. During the first twenty-four hours the bowels moved nine times; the stools were liquid, dark in color, and did not contain fat. After three days there seemed to be some improvement in her physical condition. The belly was slightly tympanitic, and she winced on palpation over the right lobe of the liver. The talkative delirium persisted, and the temperature remained 101°. The diarrhœa had now ceased, and several formed brown movements were passed. Five days after admission the belly had become soft, but the patient was stuporous, and winced when the epigastric and hepatic areas were palpated. There were a few suspicious, lenticular, rose-colored spots on the chest and bosom. Diarrhœa again set in, the stools being dark-green, and containing a considerable amount of mucus. The patient was rapidly losing flesh and strength.

At times her pulse was almost imperceptible, and the extremities and face cyanotic. The blood examined upon this day gave a positive Widal reaction. Upon the succeeding day, or the seventh in the hospital, there was slight improvement. The abdomen was softer, and a doubtful mass, several inches in diameter, and extending across the epigastric region, was felt. Upon the subsequent day the Widal test was again positive. The patient gradually sank, and died in the early morning hours of exhaustion—the tenth day in the hospital and the fifteenth day of her illness.

The autopsy was made about six hours after death. The brain and spinal cord were not examined. The body was that of a medium-sized, fairly well-nourished woman. The skin was very pale. The right heart was full of clotted blood and the ventricle flabby. The heart was normal, except a slight thickening at the base of the mitral and aortic valves. A slight degree of hypostatic congestion was present at the base of each lung. An old puckered cicatrix, probably tuberculous, was found at the apex of the right lung. Upon opening the abdomen I was at once struck by the numerous areas of fat necrosis in the omentum, and began to investigate the pancreas. A localized peritonitis existed; the smaller bowel was here covered with lymph in the epigastric region, and was matted together by rather recent fibrinous adhesions; this made a covering for a mass which was subsequently made out to be the pancreas. There was a small amount of turbid sero-sanguinous fluid in the peritoneal cavity. Through the omentum, mesentery, and mesocolon were hundreds of necrotic areas (fat necrosis), varying in size from that of a pin-point to that of a split-pea. They were yellowish-white in color, raised above the surface, with slight inflammatory areas around them. Sometimes two such spots would be confluent. When the adherent coils of the smaller bowel were raised up there was behind them a localized collection of turbid fluid, which was bounded in front by the bowel, above by the stomach, and below by the pancreas. This was unquestionably purulent exudate into the lesser peritoneal sac. The pancreas itself was enlarged to about three times its normal size, its head projecting into the duodenum. It was soft, and was with difficulty examined, because of the adhesions, and the abscess of which it was the lower boundary.

On section of the head and body of the pancreas, they were found in some areas bright red and in others coal-black in color. The tail was less affected than the body and the head. In many areas the pancreatic tissue was entirely gangrenous. There was no obstruction or calculus in the duct. The hemorrhage into the pancreas had not been large in amount, and extravasation into the surrounding tissues had not taken place. There was no inflammation or ulceration of Peyer's patches. There was no perforation of the gut. The appendix was normal. The spleen was small, soft, pliable, and intensely congested. The liver was large and fatty. The gall-bladder contained three facetted gallstones, one the size of a hickory-nut. The kidneys were fatty and contracted, the left containing a cyst the size of a large hen's egg, filled with a yellowish, slightly turbid fluid; the capsules stripped easily.

I had not, at the time of the patient's death, made a diagnosis in her case. I could see nothing upon which to base a definite opinion. The symptoms, now clear to me, were then obscured by the mental condition, and the results of the blood and urinary examination. I felt sure she had not typhoid fever; also positive that she was not uræmic, though appreciating that the kidneys were by no means normal. I was inclined to think that she had had previous intestinal obstruction, and that partial relief of the obstruction had taken place, and that we were dealing with a disappearing local peritonitis. The question of pancreatic disease was discussed, and the stools were watched, but nothing was gained from this. No tests for suppression of the pancreatic secretion were used. I could not entirely account for the unusual mental condition, and felt certain that the patient had an illness of a grave character.

Cases of acute pancreatitis are comparatively rare. Of the hemorrhagic variety Korte has collected 41, only 4 of these occurring in women. The hemorrhagic cases may be followed by suppuration of the gland—acute suppurative pancreatitis. Fitz, in his monograph in 1889, reported 22 cases, and to this list Korte has added 24. Gangrene or necrosis may follow either inflammation, hemorrhage, or suppuration of the gland. Of acute gangrenous pancreatitis Fitz has reported 15 cases, and Korte has increased this number to 40. I have gone over the records of the Pennsylvania Hospital for the past ten years (1888 to 1898), and, barring cases of primary cancer of the pancreas, of which there are a few, found no cases of acute pancreatitis recorded. There was one case (Hospital No. 1353, 1897) of pancreatic cyst in a woman, who was operated on by Dr. Thomas G. Morton. The fluid secured was yellowish; it converted starch into sugar and emulsified fat. This patient recovered. I have gone over the records of the post-mortem room since authentic records have been kept (1876), and have found, excluding cancer of the pancreas, but one additional case of acute pancreatic disease. I will give a brief history of this case:

A. F., male, aged thirty-four years, Hospital No. 714, admitted July, 1894. The patient was a rather stout young German, who used alcohol freely. Two days before admission to the hospital he complained of abdominal pain. The bowels had been moved four times on each of the previous two days. Upon admission to the hospital the man was dull; the temperature was 102.4°. It dropped the following morning to 99°, and then to normal. There was an increase of resistance, with dulness on the right side of the abdomen and tympany on the left. No mass was made out. After admission to the hospital the bowels were constipated; he vomited and retched considerably. A movement was effected with castor-oil. The urine was 1018, and contained no albumin or sugar. The man was evidently very ill, and on the third day he died, his temperature rising to 102° before death. The post-mortem showed a wide-spread fat necrosis, with the following changes in the pancreas: A hemorrhage into the gland, almost doubling its size; the pancreas was dark red in color and necrotic in areas. The changes in the gland were most marked in the head and body; the tail was but little affected. No marked pathological changes were found in the other organs.

On the other hand, however, acute pancreatitis seems to be becoming more clearly recognized or more prevalent. On December 8, 1898, before the Clinical Society of London,[5] there were reported seven cases of acute pancreatitis, and two cases of pancreatic calculi. Two other cases were also mentioned during the discussion. I know that within the last two years Dr. William Pepper had seen and diagnosed two cases of acute pancreatic disease, both of them being associated with pre-existing liver disease. I believe these cases will subsequently be reported. I have found in the report of the Presbyterian Hospital for 1898 one case of acute hemorrhagic pancreatitis. I do not find, however, that the question of acute pancreatic disease has been presented in the last decade to the College of Physicians or to the Pathological Society, though prior to 1890 Drs. Pepper, Osler and Hughes, Formad, Musser, and Starr have made contributions on that topic.

The disease is not commonly diagnosed. Dr. Osler in his last edition states that Fitz and Thayer have made the diagnosis intra vitam. I have seen the diagnosis of hemorrhagic pancreatitis made upon the operating table because of the presence of multiple areas of fat necrosis in the omentum (a case quoted by Osler), but the patient recovered, and was living four years later, when he had a recurrence. At the present time, however, cases of acute hemorrhagic pancreatitis have been sufficiently studied to enable us to form a fairly good clinical picture of the symptoms. Such cases usually occur in persons over fifty years of age, who are heavy-weights, and especially those who have used alcohol to excess.[6] The major portion of these cases have occurred in males, many of whom had a previous history of gastric and intestinal upsets. The illness begins suddenly, with severe colicky pain in the epigastric region, the symptoms usually appearing shortly after the ingestion of food, or at the beginning of the digestive process.[7] The pain does not radiate toward the left shoulder (Lancereaux); vomiting follows almost immediately. The vomited material may be copious, cosisting of partially digested food or slimy matter. It may become green or black, and sometimes contains a little liquid or clotted blood.[8] According to Lancereaux, the vomit is bilious and usually acid. Symptoms of collapse soon follow. There may be some tenderness or increased resistance over the pancreatic area. Fitz considers the presence of localized spots of tenderness over the abdomen of great diagnostic value. They are probably due to areas of fat necrosis. The temperature for the first twenty-four hours is normal or subnormal, but apt to rise after this to 100° or even to 104°. The belly usually becomes distended. The bowels are generally constipated, sometimes with alternating diarrhœa. The urine, which may be very abundant at the beginning of the attack, becomes much reduced in quantity. It may contain albumin and casts; sugar has been found. A mild delirium is sometimes present. A severe case usually terminates at the end of the third or fourth day in death. It is thus seen that these cases are similar to those of poisoning by irritant chemicals. Lancereaux cites the case of a large, fleshy woman, in whom death was preceded by convulsive movements, and in which the course was so rapid that he thought the case might have been poisoning by cyanide of potassium. Zenker compares the onset to the symptoms which follow a blow upon the pit of the stomach.

If, however, the case does not terminate fatally within the first three or four days we may be dealing with another form of pancreatic disease—namely, either the suppurative or the gangrenous. The symptoms then become those of a localized peritonitis; the belly becomes more tympanitic, the temperature slightly irregular, and the bowels may now become diarrhœic. A mild delirium is usually present. The urine is usually albuminous and lessened in quantity. Sugar has been found, and if the infection has taken place through the pancreatic ducts, and not through the blood (an important distinction according to Lancereaux), sugar is usually present. Emaciation begins and may become extreme. The abdomen may soften enough to reveal a mass on palpation in the epigastric region. Hiccough may be present, and not infrequently the patient is jaundiced. The stools, if examined, may show the presence of fat, and, if meat diet is used, an excess of undigested muscle fibre.[9] Fat may also be present in the urine (lipuria), and indican is sometimes found.

If gangrene follows acute pancreatitis, more or less of the entire gland is transformed into a gray mass with a tendency to become spongy, and to form a slough, which in some cases is attached to the abdominal wall by only a few shreds of tissue. The omental bursa (the lesser sac of the peritoneum) is not infrequently the seat of blood-stained fluid, or a large cyst in which masses of necrotic fat may be found (Fitz).

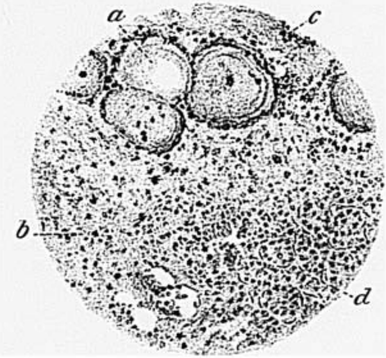
The post-mortem findings, therefore, in these cases are in brief as follows:

If hemorrhage has taken place the pancreas may be enlarged to two or three times its normal size with hemorrhagic infiltration. The blood is sometimes infiltrated into the surrounding tissues, or but a portion of the gland, usually the head or body, is involved in the hemorrhagic process. The pancreatic tissue is in areas bright red, in others black, dependent upon the duration of the hemorrhage. The cause of the hemorrhage is as yet in doubt. Fitz has not been able to discover, after careful examinations, the rupture of any bloodvessel in the gland. Lancereaux believes, however, that the bleeding takes place from miliary aneurisms, such as are frequently found in the brain and lungs of tubercular or syphilitic patients, and that have been overlooked in the examination of the pancreas Warthin,[10] who has recently studied the minute pathology of acute pancreatic disease, finds all the bloodvessels, especially the veins and capillaries, much degenerated, and considers that the hemorrhage is due to a diapedesis. The severity of the symptoms produced (pain, colic, etc.) is probably the result of pressure upon the solar plexus. The microscopical examination of these nerves has shown distinct degenerative changes. Attending this state of the pancreas, and in the inflammatory conditions, subperitoneal areas of fat necrosis, varying from the size of a pin-head to that of a pea, are always found in the omentum, mesocolon, and mesentery. This necrosis may be so excessive as to be found in the perinephritic and pericardial fat (Warthin).

Fat necrosis is always observed in the pancreas. This fat necrosis (so called by Balser) we find in this and in no other condition, though Osler states that it has been accidentally found in operations for ovarian tumors, and in lean people who have gallstones. The fat necrosis, which is so widely scattered, is generally supposed to be due to the action of the fat-splitting ferments which have been found in the necrotic areas by Flexner. Experimentally, Williams, Hildebrand, Flexner, and Hlawa have reproduced the hemorrhage and fat necrosis —Flexner by the injection of acid and alkali solution into the veins of guinea-pigs. His conclusions were that the necrosis was not produced by the acid solution used. Carnot[11] has repeated these experiments with papain and diphtheria antitoxin, and secured results identical with the above. Warthin, because of the wide-spread necrosis, the fact that bloodvessel degeneration is extreme, and that degenerated red cells and thrombi are numerous, is inclined to think the necrosis is caused by an active hæmolytic poison, the result of disturbed metabolism.

I will not enter into the minute pathology of the fat necrosis. The two illustrations which accompany the article show well the necrotic areas as they occur in both the pancreas and the omentum. The blood crystals from the hemorrhage into the pancreas show especially well, though they are of rather an unusual form, spiculated. These specimens were from sections made in the second case detailed above, and prepared at the Pennsylvania Hospital by Dr. Thomas S. Kirkbride, who kindly loaned them to me for study. The pancreatic specimen also shows, in several places, areas in which there appeared to be rather numerous bacteria, but which, upon closer inspection, are unquestionably fat crystals, the products of the fatty acids. I regret that I have not been able to illustrate this point.

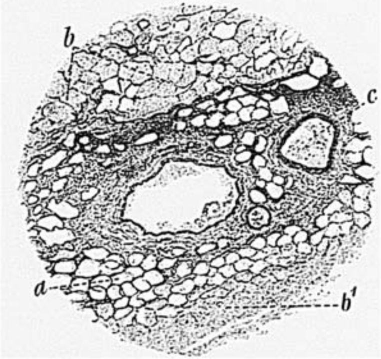
FIG. 1.



Pancreatic necrosis.

a. Necrotic fat. b. Tissue necrosis. c. Hæmatoidin crystals in spicule form. d. Acini undergoing necrosis.

FIG. 2.



Fat necrosis in omentum.

a. Normal fat. b. Necrotic fat. b^1. Total tissue necrosis. c. Artery with thrombus.

If the inflammatory process goes on to suppuration, abscess, either single or multiple, is found in the pancreas.

If gangrene of the gland occurs the pancreas becomes mushy, grayish-black, may break down altogether, and is usually the boundary of a cystic collection of fluid in the lesser peritoneal sac. Fat necrosis is found in these two conditions. If a cyst forms in the lesser peritoneal sac the case may be one of many months' duration, for the cyst may become enormous. Upon examination of the abdomen it is usually found between the stomach and the transverse colon. A very good description of these cystic formations is to be found in Korte's work.

It can, therefore, be seen from the above statements that the diagnosis of acute pancreatitis is not made with ease. It is to be differentiated from, first, acute intestinal obstruction; second, perforating gastric or duodenal ulcer; third, hepatic colic; fourth, an irritant poisoning; and, fifth, thrombosis of the mesenteric vessels.

A careful investigation into the character of the patient's previous health will always be of great assistance in the diagnosis. It is not necessary for me to enter into detail. Acute hemorrhagic pancreatitis is differentiated from acute intestinal obstruction by the severity of the onset, by the absence in the early stages of distention of the abdomen, by the tenderness in the region of the pancreas, and by the infrequency of the obstruction of the bowel in the epigastric region.

It is differentiated from ulcer of the stomach by the absence of pain immediately after eating and the absence of blood from the vomit and stool; from gallstone by the seat of pain and tenderness, and the absence of previous attacks of hepatic colic and jaundice; from irritant poison by the absence of the history of ingestion and the examination of the vomit.

Thrombosis of the mesenteric vessels is rare. In the majority of cases the heart or abdominal aorta is diseased; there is usually incessant vomiting and diarrhœa, with rapid abdominal distention.

The prognosis of these cases is usually extremely grave; the majority of them have resulted fatally. There have been reported four or five recoveries after operation—one by Osler, before mentioned, one by Korte, one by Thayer, one by J. Langton, and one by Manges.[12] Manges' case illustrates very well the onset of pain after the ingestion of food, and shows that an exploratory laparotomy alone is of such assistance that recovery may take place. Troyfoyer's well-known case was living seventeen years after the pancreas had been passed by the bowel.

The best treatment, therefore, for acute pancreatitis consists in making a correct diagnosis and handing the patient over to the care of the surgeon. I believe the next few years will present more correct diagnoses, with a better percentage of recoveries from operation.[13]

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THE DIAGNOSTIC VALUE OF THE WINTRICH TRACHEAL SOUND.

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MODERATE percussion over any portion of the normal thorax gives a sound of a certain character which is not modified by the patient holding the mouth open. If at the same time the examiner percusses the thorax he place his ear near the patient's nose he will perceive no difference in the character of the sound. Under normal conditions moderate percussion-vibrations are not communicated to the large bronchi, consequently the column of air in the trachea is not implicated in the percussion, and no sound issues from the nose.

Under certain pathological conditions the vibrations of a moderate percussion stroke will be communicated to the large bronchi, thus involving the column of air in the trachea. Under such conditions there will be two sounds conducted to the examiner's ear—one directly from the thorax through the atmosphere, the other through the trachea and nares or open mouth. Wintrich described the modification of the percussion note over cavities in the lung that occurred when the patient held the mouth alternately open and closed. When the mouth was opened the percussion-note became more tympanitic and clearer and higher in pitch, because the open mouth permits the pharynx to act as a resonator over the column of air in the trachea, which is set in vibration when the percussed cavity communicates with a bronchus. Williams noticed the same modification of the percussion-note over consolidated areas of the lung at the apices and also over the upper thorax when the lung was compressed from pleurisy with effusion. In both consolidation and compression of the upper lobes of the lungs the consistency of the lung is increased, so that the vibrations from moderate